REMARKS

These remarks are in response to the Office Action mailed July 14, 2003. Claims 1 to 86 are pending. Claims 1 to 80 stand withdrawn from consideration as drawn to an unelected species. Claims 81 to 86 are under consideration.

Applicants thank the Examiner for the interview held September 23, 2003. Applicants believe that the amendments to the specification and remarks below address all remaining rejections and the issues raised by the Examiner during the interview. Applicants respectfully request reconsideration of the present application.

Regarding the Amendments to the Specification

The specification has been amended to correct several typographical errors. In particular, the specification has been amended at pages 38 and 40 to provide the correct sequences and sequence identifiers for S216, which is "LYRSPSMPENL" and corresponds to SEQ ID NO:3, and S216A, which is "LYRSPAMPENL" and corresponds to SEQ ID NO:1897. Support for the amendment to the sequence of S216 as LYRSPSMPENL can be found throughout the specification, for example, at page 21, lines 42-43, which discloses the human Cdc25C sequence, amino acids 211 to 221 (LYRSPSMPENL), from which S216 was derived. Support for the amendment to the sequence of S216A as LYRSPAMPENL can be found, for example, at page 99, Table 2. Thus, as the amendments to correct typographical errors are supported by the specification, no new matter is added and entry thereof is respectfully requested.

REJECTION UNDER 35 U.S.C. §112

The rejection of claims 81 to 86 under 35 U.S.C. §112, first paragraph as allegedly lacking enablement is respectfully traversed. The Patent Office indicates that allegedly the specification "does not disclose whether the polypeptide of SEQ ID NO:1897, or any other peptide comprising SEQ ID NO:1897 is capable of acting as a substrate of Chk2." The Examiner asserts that "because the specification does not provide guidance supporting the assertion that a polypeptide of SEQ ID NO:1897 would be expected to act as a substrate of Chk2, the skilled artisan could not use the claimed invention without a reasonable expectation of success." [Office Action, page 3]

Applicants first wish to point out that elected sequence denoted as SEQ ID NO:1897 (LYRSPAMPENL) is identical to SEQ ID NO:1924 (LYRSPAMPENL) in the subject application. For example, the specification discloses SEQ ID NO:1897 as LYRSPAMPENL at page 3, line 25, and in claim 32; and SEQ ID NO:1924 as S216A (LYRSPAMPENL) at page 99 (Table 2). Thus, SEQ ID NO:1897 and SEQ ID NO:1924 are identical and refer to LYRSPAMPENL (S216A). TAT-S216A (SEQ ID NO:1933, see page 9, lines 25-27) is a fusion of an eleven amino acid HIV TAT sequence (TGRKKRRQRRR, SEQ ID NO:1899, see page 38, lines 8-12) and S216A.

The specification adequately enables claims 81 to 86. The specification teaches a peptide, SEQ ID NO:1897 (LYRSPAMPENL), that can inhibit or abrogate G2 checkpoint arrest without being phosphorylated by Chk1 or Chk2 kinases. For example, the specification discloses that TAT-S216A inhibits phosphorylation of Cdc25 by hChk1, but is not itself phosphorylated by hChk1 (page 40, line 27, to page 41, line 6). The specification also discloses that G2 arrest is abrogated by TAT-S216A in response to bleomycin, gamma-irradiation and cisplatin (page 41, lines 21-28; see, also, Fig. 2A). Thus, TAT-S216A can abrogate or inhibit G2 arrest without acting as a phosphorylation substrate of hChk1.

Accordingly, as the specification teaches peptides that can inhibit or abrogate G2 checkpoint arrest that need not be phosphorylated, and furthermore, exemplifies such a peptide (SEQ ID NO:1897, LYRSPAMPENL) that inhibits or abrogates G2 checkpoint arrest, undue experimentation would not be required to practice claims 81 to 86. Thus, as claims 81 to 86 can be practiced without undue experimentation, the claims are adequately enabled. Accordingly, Applicants respectfully request that the rejection under 35 U.S.C. §112, first paragraph be withdrawn.

CONCLUSION

In summary, for the reasons set forth herein, Applicants maintain that claims 81 to 86 clearly and patentably define the invention, respectfully request that the Examiner reconsider the grounds set forth in the Office Action, and respectfully request the allowance of the claims which are now pending.

If the Examiner would like to discuss any of the issues raised in the Office Action, Applicant's representative can be reached at (858) 509-4065.

Please charge any additional fees, or make any credits, to Deposit Account No. 03-3975.

Respectfully submitted,

Date: 10-7-03

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COPY OF CLAIMS:

Claims 1 – 80: (Withdrawn)

81. (Previously presented.) A method for screening for compounds capable of specifically inhibiting or abrogating the G2 cell cycle arrest checkpoint comprising the following steps

(a) providing a test compound and a polypeptide as set forth in claim 1 or claim 57;

(b) providing a G1 checkpoint impaired cell;

(c) contacting the cell of step (b) with the test compound or the polypeptide of step (a) and a DNA damaging treatment or an M phase checkpoint activator; and

(d) measuring the amount of DNA in the cells after the contacting of step (c) to determine if the test compound has inhibited or abrogated the G2 cell cycle arrest checkpoint, wherein the polypeptide of step (a) acts as a G2-checkpoint-inhibiting positive control.

82. (Previously presented.) The method of claim 81, wherein the amount of DNA is measured using propidium iodide and FACS analysis.

83. (Previously presented.) The method of claim 81, wherein the amount of DNA is measured after about 10 to about 72 hours after the contacting of step (c).

84. (Previously presented.) The method of claim 81, wherein the cell is contacted with an M phase checkpoint activator and a test compound or a polypeptide of step (a), wherein a test compound that has not inhibited or abrogated the arrest at the M phase checkpoint of the cell cycle after contacting the cell with an M phase activator is a specific inhibitor of the G2 cell cycle arrest checkpoint.

85. (Previously presented.) The method of claim 84, wherein the M phase checkpoint activator is colchicine or nocodazole.

86. (Previously presented.) The method of claim 81, wherein the DNA damaging treatment is 5-fluorouracil (5-FU), rebeccamycin, adriamycin, bleomycin, cisplatin, hyperthermia, UV irradiation or gamma-irradiation.